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=> S  (raf-1 (3A) RBD) (8A) (variant or mutant or mutated or mutation or mutating
or mutagenesis or substitution or substitute or substituted)
L1            2 (RAF-1 (3A) RBD) (8A) (VARIANT OR MUTANT OR MUTATED OR MUTATION
                OR MUTATING OR MUTAGENESIS OR SUBSTITUTION OR SUBSTITUTE OR SUBS
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=> S (gtpas or ras) (8A) (binding affinity)
L2           66 (GTPAS OR RAS) (8A) (BINDING AFFINITY)

=> s l1 and l2
L3            0 L1 AND L2

=> d l1 1-2 bib ab

L1   ANSWER 1 OF 2  EMBASE  COPYRIGHT (c) 2008 Elsevier B.V. All rights
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AN   1999061407  EMBASE
TI   Nuclear magnetic resonance and molecular dynamics studies on the
     interactions of the Ras-binding domain of Raf-1 with wild-type and mutant
     Ras proteins.
AU   Tateno, Masaru; Ebisuzaki, Toshikazu
CS   Computational Science Laboratory, Inst. Phys. and Chem. Res. (RIKEN), 2-1
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SO Journal of Molecular Biology, (12 Feb 1999) Vol. 286, No. 1, pp. 219-232. Refs: 74
ISSN: 0022-2836 CODEN: JMOBAK
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LA English
SL English
ED Entered STN: 25 Feb 1999
Last Updated on STN: 25 Feb 1999
AB The Ras protein and its homolog, Rap1A, have an identical 'effector region' (residues 32-40) preceded by Asp30-Glu31 and Glu30-Lys31, respectively. In the complex of the 'Ras-like' E30D/K31E mutant Rap1A with the Ras-binding domain (RBD), residues 51-131 of Raf-1, Glu31 in Rap1A forms a tight salt bridge with Lys84 in Raf-1. However, we have recently found that Raf-1 RBD binding of Ras is indeed reduced by the E31K mutation, but is not affected by the E31A mutation. Here, the 'Rap1A-like' D30E/E31K mutant of Ras was prepared and shown to bind the Raf-1 RBD less strongly than wild-type Ras, but slightly more tightly than the E31K mutant. The backbone (1)H, (13)C, and (15)N magnetic resonances of the Raf-1 RBD were assigned in complexes with the wild-type and D30E/E31K mutant Ras proteins in the guanosine 5'-O-(β,γ-imidotriphosphate)-bound form. The Lys84 residue in the Raf-1 RBD exhibited a large change in chemical shift upon binding wild-type Ras, suggesting that Lys84 interacts with wild-type Ras. The D30E/E31K mutant of Ras caused nearly the same perturbations in Raf-1 chemical shifts, including that of Lys84. We hypothesized that Glu31 in Ras may not be the major salt bridge partner of Lys84 in Raf-1. A molecular dynamics simulation of a model structure of the Raf-1 RBD.ovrhdot.Ras.ovrhdot.GTP complex suggested that Lys84 in Raf-1 might instead form a tight salt bridge with Asp33 in Ras. Consistent with this, the D33A mutation in Ras greatly reduced its Raf-1 RBD binding activity. We conclude that the major salt bridge partner of Lys84 in Raf-1 may be Asp33 in Ras.

L1 ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1999:134549 BIOSIS
DN PREV199900134549
TI Nuclear magnetic resonance and molecular dynamics studies on the interactions of the Ras-binding domain of Raf-1 with wild-type and mutant Ras proteins.
AU Terada, Tohru; Ito, Yutaka; Shirouzu, Mikako; Tateno, Masaru; Hashimoto, Kyoko; Kigawa, Takanori; Ebisuzaki, Toshikazu; Takio, Koji; Shibata, Takehiko; Yokoyama, Shigeyuki [Reprint author]; Smith, Brian O.; Laue, Ernest D.; Cooper, Jonathan A.
CS Cellular Signaling Lab., Inst. Phys. Chem. Res., 2-1 Hirosawa, Wako-shi, Saitama 351-0198, Japan
SO Journal of Molecular Biology, (Feb. 12, 1999) Vol. 286, No. 1, pp. 219-232. print.

CODEN: JMOBAK. ISSN: 0022-2836.
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LA English
ED Entered STN: 31 Mar 1999
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AB The Ras protein and its homolog, Rap1A, have an identical "effector region" (residues 32-40) preceded by Asp3O-Glu31 and Glu3O-Lys31, respectively. In the complex of the "Ras-like" E30D/K31E mutant RaplA with the Ras-binding domain (RBD), residues 51-131 of Raf-l, Glu3l in RaplA forms a tight salt bridge with Lys84 in Raf-1. However, we have recently found that Raf-I RBD binding of Ras is indeed reduced by the E31K mutation, but is not affected by the E31A mutation. Here, the "RaplA-like" D30E/E31K mutant of Ras was prepared and shown to bind the Raf-1 RBD less strongly than wild-type Ras, but slightly more tightly than the E31K mutant. The backbone 1H, 13C, and 15N magnetic resonances of the Raf-1 RBD were assigned in complexes with the wild-type and D30E/E31K mutant Ras proteins in the guanosine 5'-O-(beta,gamma-imidotriphosphate)-bound form. The Lys84 residue in the Raf-1 RBD exhibited a large change in chemical shift upon binding wild-type Ras, suggesting that Lys84 interacts with wild-type Ras. The D30E/E31K mutant of Ras caused nearly the same perturbations in Raf-1 chemical shifts, including that of Lys84. We hypothesized that Glu3l in Ras may not be the major salt bridge partner of Lys84 in Raf-1. A molecular dynamics simulation of a model structure of the Raf-1 RBDcntdotRascntdotGTP complex suggested that Lys84 in Raf-1 might instead form a tight salt bridge with Asp33 in Ras. Consistent with this, the D33A mutation in Ras greatly reduced its Raf -1 RBD binding activity. We conclude that the major salt bridge partner of Lys84 in Raf-1 may be Asp33 in Ras.